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2600 MCQ with Illustrated Answer
Full Topics Note
14 Chapter

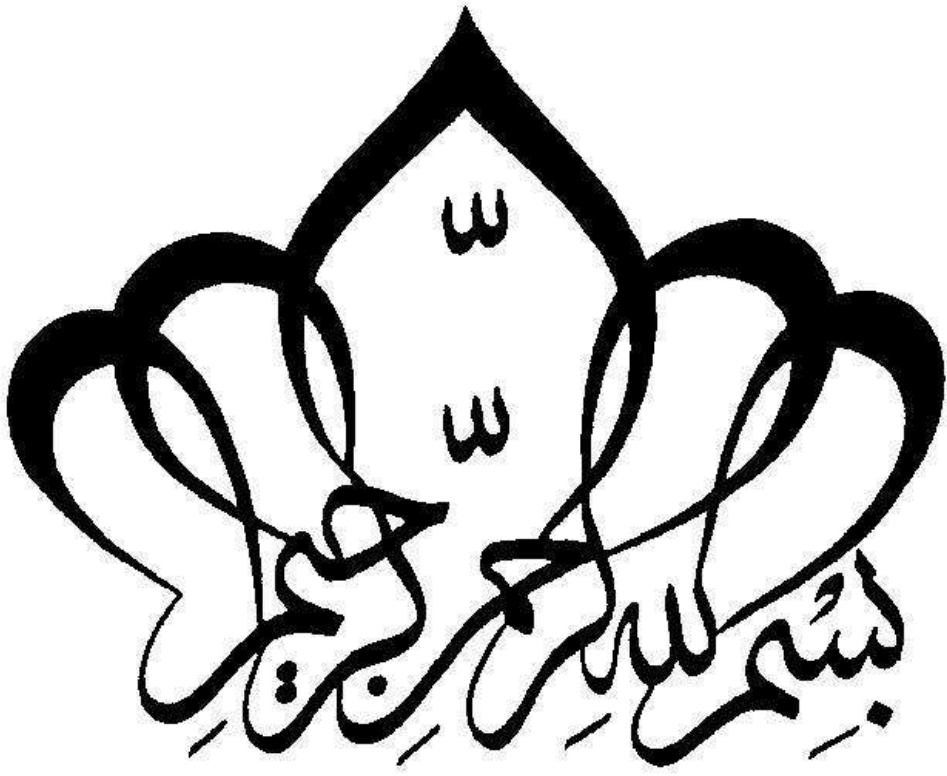
Dermatology

P. M MCQ Bank 2017



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قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا
إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ

إهداء

للكل آية من كتاب الله حفظناها ولكل معلومة قرأناها ونسيناها لعل الله يحمينا من النسيان ويروها إلينا
للكل شخص وعونا له في ظهر الغيب لعل الله أن يستجيب وعائنا
للكل مريض قرأله ما أله لعل الله يشفيه ويعير إليه صحته وعافيته
للكل لحظة تألم بها أحببنا لعل الله لا يريهم الألم ولا يضييهم مرة أخرى
للكل موقف اجتجت به عينا فلم تلباني لعل الله لا يضرنني بهما في حياتي
للكل حالم صاحب أهراء ومباوئ لعل الله يحقق له مقاصده حتى يرضيه ويرضى عنه
للكل مجتهد لعل الله يجعل له من التوفيق أوفر حظ ونصيب ويهون له الأسباب كلها من حيث لا يدري من غير ضرر ومضرة ولا فتنة مضله
للكل من فقر شيئا في الحياة لعل الله يعيره إليه أو يعوضه بما يسعده
للكل شخص ابتعدنا عنه لعل الله يجمعه بمن هم أفضل منا
وأخيرا وليس آخرا لأهلنا لعل الله يجعلها لهم صرقة جارية ترو إليهم قليلا من فضلهم علينا وتكون سببا يلم شملنا تحت سقف واحد بعد أن
هجرتنا الحروب والمصائب

يقول أحد الصالحين

إِذَا رَأَيْتَ أَنَّ اللَّهَ وَفَقَكَ لِإِخْوَةِ تَعْيْنِكَ عَلَى الْخَيْرِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ بِكَ خَيْرًا !! وَإِذَا وَفَقَكَ اللَّهَ لِلدُّعَاءِ.. فَأَعْلَمْ أَنَّ اللَّهَ يَرِيدُ أَنْ يُعْطِيَكَ !! وَإِذَا
وَفَقَكَ لِقَضَاءِ حَوَائِجِ النَّاسِ فَأَعْلَمْ أَنَّ لَكَ يَتَخَلَّى عَنْكَ !! وَإِذَا وَفَقَكَ اللَّهَ لِذِكْرِهِ.. فَأَعْلَمْ أَنَّكَ مُحِبٌّ !!.. وَإِذَا أَحْبَبَّكَ أَحَدٌ.. وَنَصَرَكَ.. وَأَيَّرَكَ..
وَأُسْتَجَابَ لِدُعَائِكَ !!

This issue of **MRCPPART1 MADE EASY 2017** contains the following:

- Full topic note.
- Around 2600 MCQs from **PM 2017 BANK** covering 14 chapter in internal medicine
- External links for the related guidelines (such as NICE, SIGN, RCP ...etc).
- Comments with illustrative pictures and links to external media.
- Most Recommended QBank for MRCP Part 1, Arab Board, Jordanian Board and other internal medicine assessment exams.

Please Note that

Each chapter begins with notes section. These notes are repeated under the Questions in the website. In these collections the notes are not repeated under each question except in some to avoid unnecessary repetition. For clarity and convenience the title of the related note is only mentioned after the comments in case the reader want to refer back to the respective note in the notes section.

The illustration below is to show and Example

☐	Minimal change disease
☐	Post-streptococcal glomerulonephritis
☐	Goodpasture's syndrome

Goodpasture's syndrome

- IgG deposits on renal biopsy
- anti-GBM antibodies

• These changes are characteristic of Goodpasture's syndrome.

[Discuss and give feedback](#)

Goodpasture's syndrome

Comment of the Q

title of the note in case the reader want to refer back to the notes sections

MRCPPART1 MADE EASY 2017

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إعداد وتنسيق

M. HABAYEB

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&

A. MURAD

لا تنسونا من صالح دعائكم

ما كان من خطأ فمن أنفسنا وما كان من توفيق فمن الله

تم بحمد الله وتوفيقه ومنه

جعل الله عملنا متقبلاً خالصاً لوجهه الكريم

Reference ranges

Reference ranges vary according to individual labs. All values are for adults unless otherwise stated

Full blood count

Haemoglobin	Men: 13.5-18 g/dl Women: 11.5-16 g/dl
Mean cell volume	82-100 fl
Platelets	150-400 * 10 ⁹ /l
White blood cells	4-11 * 10 ⁹ /l

Urea and electrolytes

Sodium	135-145 mmol/l
Potassium	3.5 - 5.0 mmol/l
Urea	2.0-7 mmol/l
Creatinine	55-120 umol/l
Bicarbonate	22-28 mmol/l
Chloride	95-105 mmol/l

Liver function tests

Bilirubin	3-17 umol/l
Alanine transferase (ALT)	3-40 iu/l
Aspartate transaminase (AST)	3-30 iu/l
Alkaline phosphatase (ALP)	30-100 umol/l
Gamma glutamyl transferase (γGT)	8-60 u/l
Albumin	35-50 g/l
Total protein	60-80 g/l

Other haematology

Erythrocyte sedimentation rate (ESR)	Men: < (age / 2) mm/hr Women: < ((age + 10) / 2) mm/hr
Prothrombin time (PT)	10-14 secs

Activated partial thromboplastin time (APTT)	25-35 secs
Ferritin	20-230 ng/ml
Vitamin B12	200-900 ng/l
Folate	3.0 nmol/l
Reticulocytes	0.5-1.5%
D-Dimer	< 400 ng/ml

Other biochemistry

Calcium	2.1-2.6 mmol/l
Phosphate	0.8-1.4 mmol/l
CRP	< 10 mg/l
Thyroid stimulating hormone (TSH)	0.5-5.5 mu/l
Free thyroxine (T4)	9-18 pmol/l
Total thyroxine (T4)	70-140 nmol/l
Amylase	70-300 u/l
Uric acid	0.18-0.48 mmol/l

Arterial blood gases

pH	7.35 - 7.45
pCO2	4.5 - 6.0 kPa
pO2	10 - 14 kPa

Lipids

Desirable lipid values depend on other risk factors for cardiovascular disease, below is just a guide:

Total cholesterol	< 5 mmol/l
Triglycerides	< 2 mmol/l
HDL cholesterol	> 1 mmol/l
LDL cholesterol	< 3 mmol/l

Chapter: Dermatology

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Acanthosis nigricans

Describes symmetrical, brown, velvety plaques that are often found on the neck, axilla and groin

Causes

- gastrointestinal cancer
- diabetes mellitus
- obesity
- polycystic ovarian syndrome
- acromegaly
- Cushing's disease
- hypothyroidism
- familial
- Prader-Willi syndrome
- drugs: oral contraceptive pill, nicotinic acid



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External Links

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Acanthosis nigricans

Acne rosacea

Acne rosacea is a chronic skin disease of unknown aetiology

Features

- typically affects nose, cheeks and forehead
- flushing is often first symptom
- telangiectasia are common
- later develops into persistent erythema with papules and pustules
- rhinophyma
- ocular involvement: blepharitis

Management

- topical metronidazole may be used for mild symptoms (i.e. Limited number of papules and pustules, no plaques)
- more severe disease is treated with systemic antibiotics e.g. Oxytetracycline
- recommend daily application of a high-factor sunscreen
- camouflage creams may help conceal redness
- laser therapy may be appropriate for patients with prominent telangiectasia

External Links

[BNF](#)

Acne rosacea

[Clinical Knowledge Summaries](#)

Acne rosacea guideline

[Dermnet NZ](#)

Acne rosacea

Acne vulgaris

Acne vulgaris is a common skin disorder which usually occurs in adolescence. It typically affects the face, neck and upper trunk and is characterised by the obstruction of the pilosebaceous follicle with keratin plugs which results in comedones, inflammation and pustules.

Epidemiology

- affects around 80-90% of teenagers, 60% of whom seek medical advice
- acne may also persist beyond adolescence, with 10-15% of females and 5% of males over 25 years old being affected

Pathophysiology is multifactorial

- follicular epidermal hyperproliferation resulting in the formation of a keratin plug. This in turn causes obstruction of the pilosebaceous follicle. Activity of sebaceous glands may be controlled by androgen, although levels are often normal in patients with acne
- colonisation by the anaerobic bacterium *Propionibacterium acnes*
- inflammation

External Links

[Clinical Knowledge Summaries](#)

Acne vulgaris guidelines

Acne vulgaris: management

Acne vulgaris is a common skin disorder which usually occurs in adolescence. It typically affects the face, neck and upper trunk and is characterised by the obstruction of the pilosebaceous follicles with keratin plugs which results in comedones, inflammation and pustules.

Acne may be classified into mild, moderate or severe:

- mild: open and closed comedones with or without sparse inflammatory lesions
- moderate acne: widespread non-inflammatory lesions and numerous papules and pustules
- severe acne: extensive inflammatory lesions, which may include nodules, pitting, and scarring.

A simple step-up management scheme often used in the treatment of acne is as follows:

- single topical therapy (topical retinoids, benzyl peroxide)
- topical combination therapy (topical antibiotic, benzoyl peroxide, topical retinoid)
- oral antibiotics: e.g. Oxytetracycline, doxycycline. Improvement may not be seen for 3-4 months. Minocycline is now considered less appropriate due to the possibility of irreversible pigmentation. Gram negative folliculitis may occur as a complication of long-term antibiotic use - high-dose oral trimethoprim is effective if this occurs
- oral isotretinoin: only under specialist supervision

There is no role for dietary modification in patients with acne

External Links

[Clinical Knowledge Summaries](#)

Acne vulgaris guidelines

Actinic keratoses

Actinic, or solar, keratoses (AK) is a common premalignant skin lesion that develops as a consequence of chronic sun exposure

Features

- small, crusty or scaly, lesions
- may be pink, red, brown or the same colour as the skin
- typically on sun-exposed areas e.g. temples of head
- multiple lesions may be present

Management options include

- prevention of further risk: e.g. sun avoidance, sun cream
- fluorouracil cream: typically a 2 to 3 week course. The skin will become red and inflamed - sometimes topical hydrocortisone is given following fluorouracil to help settle the inflammation
- topical diclofenac: may be used for mild AKs. Moderate efficacy but much fewer side-effects
- topical imiquimod: trials have shown good efficacy
- cryotherapy
- curettage and cautery

External Links

[British Association of Dermatologists](#)

2007 Actinic keratoses guidelines

[DermNet NZ](#)

Actinic keratoses

Allergy tests

Skin prick test	Most commonly used test as easy to perform and inexpensive. Drops of diluted allergen are placed on the skin after which the skin is pierced using a needle. A large number of allergens can be tested in one session. Normally includes a histamine (positive) and sterile water (negative) control. A wheal will typically develop if a patient has an allergy. Can be interpreted after 15 minutes Useful for food allergies and also pollen
Radioallergosorbent test (RAST)	Determines the amount of IgE that reacts specifically with suspected or known allergens, for example IgE to egg protein. Results are given in grades from 0 (negative) to 6 (strongly positive) Useful for food allergies, inhaled allergens (e.g. Pollen) and wasp/bee venom Blood tests may be used when skin prick tests are not suitable, for example if there is extensive eczema or if the patient is taking antihistamines
Skin patch testing	Useful for contact dermatitis. Around 30-40 allergens are placed on the back. Irritants may also be tested for. The patches are removed 48 hours later with the results being read by a dermatologist after a further 48 hours

External Links

[NICE](#)

2011 Food allergy in children and young people

Alopecia

Alopecia may be divided into scarring (destruction of hair follicle) and non-scarring (preservation of hair follicle)

Scarring alopecia

- trauma, burns
- radiotherapy
- lichen planus
- discoid lupus
- tinea capitis*

Non-scarring alopecia

- male-pattern baldness
- **drugs:** cytotoxic drugs, carbimazole, heparin, oral contraceptive pill, colchicine
- **nutritional:** iron and zinc deficiency
- **autoimmune:** alopecia areata
- telogen effluvium (hair loss following stressful period e.g. surgery)
- trichotillomania

*scarring may develop in untreated tinea capitis if a kerion develops.

Alopecia areata

Alopecia areata is a presumed autoimmune condition causing localised, well demarcated patches of hair loss. At the edge of the hair loss, there may be small, broken 'exclamation mark' hairs

Hair will regrow in 50% of patients by 1 year, and in 80-90% eventually. Careful explanation is therefore sufficient in many patients. **Other treatment options include:**

- topical or intralesional corticosteroids
- topical minoxidil
- phototherapy
- dithranol
- contact immunotherapy
- wigs

External Links

[British Association of Dermatologists](#)

Alopecia areata guidelines

[Clinical Knowledge Summaries](#)

Alopecia areata guidelines

Basal cell carcinoma

Basal cell carcinoma (BCC) is one of the three main types of skin cancer. Lesions are also known as rodent ulcers and are characterised by slow-growth and local invasion. Metastases are extremely rare. BCC is the most common type of cancer in the Western world.

Features

- many types of BCC are described. The most common type is nodular BCC, which is described here
- sun-exposed sites, especially the head and neck account for the majority of lesions
- initially a pearly, flesh-coloured papule with telangiectasia
- may later ulcerate leaving a central 'crater'

Management options:

- surgical removal
- curettage
- cryotherapy
- topical cream: imiquimod, fluorouracil
- radiotherapy





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External Links

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Basal cell carcinoma

Bullous pemphigoid

Bullous pemphigoid is an autoimmune condition causing sub-epidermal blistering of the skin. This is secondary to the development of antibodies against hemidesmosomal proteins BP180 and BP230

Bullous pemphigoid is more common in elderly patients. **Features include**

- itchy, tense blisters typically around flexures
- the blisters usually heal without scarring
- mouth is usually spared*

Skin biopsy

- immunofluorescence shows IgG and C3 at the dermoepidermal junction

Management

- referral to dermatologist for biopsy and confirmation of diagnosis
- oral corticosteroids are the mainstay of treatment
- topical corticosteroids, immunosuppressants and antibiotics are also used



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*in reality around 10-50% of patients have a degree of mucosal involvement. It would however be unusual for an exam question to mention mucosal involvement as it is seen as a classic differentiating feature between pemphigoid and pemphigus.

External Links

[DermNet NZ](https://www.dermnetnz.org/)

Bullous pemphigoid

[British Association of Dermatologists](https://www.britishtoderm.org/)

Bullous pemphigoid guidelines

Contact dermatitis

There are two main types of contact dermatitis

- irritant contact dermatitis: common - non-allergic reaction due to weak acids or alkalis (e.g. detergents). Often seen on the hands. Erythema is typical, crusting and vesicles are rare
- allergic contact dermatitis: type IV hypersensitivity reaction. Uncommon - often seen on the head following hair dyes. Presents as an acute weeping eczema which predominately affects the margins of the hairline rather than the hairy scalp itself. Topical treatment with a potent steroid is indicated

Cement is a frequent cause of contact dermatitis. The alkaline nature of cement may cause an irritant contact dermatitis whilst the dichromates in cement also can cause an allergic contact dermatitis

Dermatitis herpetiformis

Dermatitis herpetiformis is an autoimmune blistering skin disorder associated with coeliac disease. It is caused by deposition of IgA in the dermis.

Features

- itchy, vesicular skin lesions on the extensor surfaces (e.g. elbows, knees, buttocks)

Diagnosis

- skin biopsy: direct immunofluorescence shows deposition of IgA in a granular pattern in the upper dermis

Management

- gluten-free diet
- dapsone



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Dermatitis herpetiformis

Eczema herpeticum

Eczema herpeticum describes a severe primary infection of the skin by herpes simplex virus 1 or 2. It is more commonly seen in children with atopic eczema. As it is potentially life threatening children should be admitted for IV aciclovir

External Links

[DermNet NZ](https://www.dermnetnz.org/)

Eczema herpeticum

Eczema: topical steroids

Use weakest steroid cream which controls patients symptoms

The table below shows topical steroids by potency:

Mild	Moderate	Potent	Very potent
Hydrocortisone 0.5-2.5%	Betamethasone valerate 0.025% (Betnovate RD) Clobetasone butyrate 0.05% (Eumovate)	Fluticasone propionate 0.05% (Cutivate) Betamethasone valerate 0.1% (Betnovate)	Clobetasol propionate 0.05% (Dermovate)

Finger tip rule

- 1 finger tip unit (FTU) = 0.5 g, sufficient to treat a skin area about twice that of the flat of an adult hand

Topical steroid doses for eczema in adults:

Area of skin	Fingertip units per dose
Hand and fingers (front and back)	1.0
A foot (all over)	2.0
Front of chest and abdomen	7.0
Back and buttocks	7.0
Face and neck	2.5
An entire arm and hand	4.0
An entire leg and foot	8.0

The BNF makes recommendation on the quantity of topical steroids that should be prescribed for an adult for a single daily application for 2 weeks:

Area	Amount
Face and neck	15 to 30 g
Both hands	15 to 30 g
Scalp	15 to 30 g
Both arms	30 to 60 g
Both legs	100 g
Trunk	100 g
Groin and genitalia	15 to 30 g

External Links

[British Association of Dermatologists](#)

Atopic eczema guidelines

Erythema ab igne

Erythema ab igne is a skin disorder caused by over exposure to infrared radiation. Characteristic features include reticulated, erythematous patches with hyperpigmentation and telangiectasia. A typical history would be an elderly women who always sits next to an open fire.

If the cause is not treated then patients may go on to develop squamous cell skin cancer.



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Erythema ab igne



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Erythema ab igne

External Links

[DermNet NZ](https://www.dermnetnz.org/)

Erythema ab igne

Erythema multiforme

Features

- target lesions
- initially seen on the back of the hands / feet before spreading to the torso
- upper limbs are more commonly affected than the lower limbs
- pruritus is occasionally seen and is usually mild

If symptoms are severe and involve blistering and mucosal involvement the term Stevens-Johnson syndrome is used.

Causes

- **viruses:** herpes simplex virus (the most common cause), Orf*
- **idiopathic**
- **bacteria:** *Mycoplasma*, *Streptococcus*
- **drugs:** penicillin, sulphonamides, carbamazepine, allopurinol, NSAIDs, oral contraceptive pill, nevirapine
- **connective tissue disease** e.g. Systemic lupus erythematosus
- **sarcoidosis**
- **malignancy**



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*Orf is a skin disease of sheep and goats caused by a parapox virus

External Links

[DermNet NZ](https://www.dermnetnz.org/)

Erythema multiforme

Erythema nodosum

Overview

- inflammation of subcutaneous fat
- typically causes tender, erythematous, nodular lesions
- usually occurs over shins, may also occur elsewhere (e.g. forearms, thighs)
- usually resolves within 6 weeks
- lesions heal without scarring

Causes

- infection: streptococci, TB, brucellosis
- systemic disease: sarcoidosis, inflammatory bowel disease, Behcet's
- malignancy/lymphoma
- drugs: penicillins, sulphonamides, combined oral contraceptive pill
- pregnancy



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Erythema nodosum

Erythrasma

Erythrasma is a generally asymptomatic, flat, slightly scaly, pink or brown rash usually found in the groin or axillae. It is caused by an overgrowth of the diphtheroid *Corynebacterium minutissimum*

Examination with Wood's light reveals a coral-red fluorescence.

Topical miconazole or antibacterial are usually effective. Oral erythromycin may be used for more extensive infection

External Links

[DermNet NZ](#)

Erythrasma

Erythroderma

Erythroderma is a term used when more than 95% of the skin is involved in a rash of any kind

Causes of erythroderma

- eczema
- psoriasis
- drugs e.g. gold
- lymphoma, leukaemia
- idiopathic

Erythrodermic psoriasis

- may result from progression of chronic disease to an exfoliative phase with plaques covering most of the body. Associated with mild systemic upset
- more serious form is an acute deterioration. This may be triggered by a variety of factors such as withdrawal of systemic steroids. Patients need to be admitted to hospital for management



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This image shows the generalised erythematous rash seen in patients with erythroderma, sometimes referred to as '**red man syndrome**'



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Note the extensive exfoliation seen in this patient

Fungal nail infections

Onychomycosis is fungal infection of the nails. **This may be caused by**

- dermatophytes - mainly *Trichophyton rubrum*, accounts for 90% of cases
- yeasts - such as *Candida*
- non-dermatophyte moulds

Features

- 'unsightly' nails are a common reason for presentation
- thickened, rough, opaque nails are the most common finding

Investigation

- nail clippings
- scrapings of the affected nail

Management

- **treatment** is successful in around 50-80% of people
- **diagnosis** should be confirmed by microbiology before starting treatment.

Chapter: Dermatology

- **dermatophyte infection:** oral terbinafine is currently recommended first-line with oral itraconazole as an alternative. Six weeks - 3 months therapy is needed for fingernail infections whilst toenails should be treated for 3 - 6 months
- **Candida infection:** mild disease should be treated with topical antifungals (e.g. Amorolfine) whilst more severe infections should be treated with oral itraconazole for a period of 12 weeks

External Links

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Fungal nail infections

Granuloma annulare

Basics

- papular lesions that are often slightly hyperpigmented and depressed centrally
- typically occur on the dorsal surfaces of the hands and feet, and on the extensor aspects of the arms and legs

A number of associations have been proposed to conditions such as diabetes mellitus but there is only weak evidence for this

External Links

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Granuloma annulare

Herpes simplex virus

There are two strains of the herpes simplex virus (HSV) in humans: HSV-1 and HSV-2. Whilst it was previously thought HSV-1 accounted for oral lesions (cold sores) and HSV-2 for genital herpes it is now known there is considerable overlap

Features

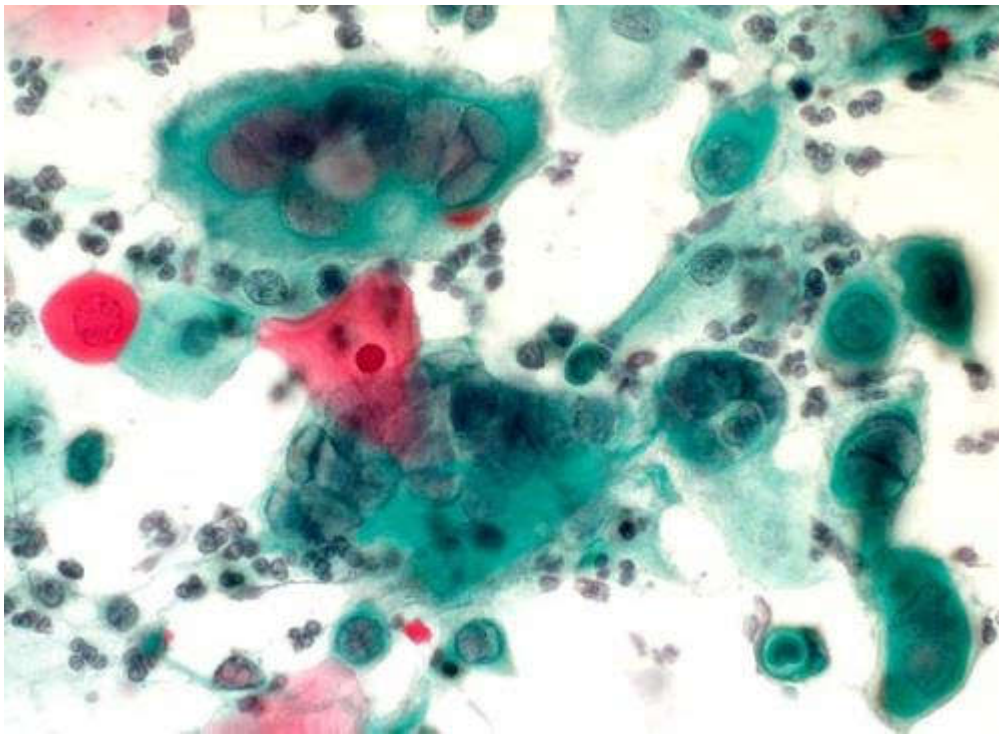
- primary infection: may present with a severe gingivostomatitis
- cold sores
- painful genital ulceration

Management

- gingivostomatitis: oral aciclovir, chlorhexidine mouthwash
- cold sores: topical aciclovir although the evidence base for this is modest
- genital herpes: oral aciclovir. Some patients with frequent exacerbations may benefit from longer term aciclovir

Pregnancy

- elective caesarean section at term is advised if a primary attack of herpes occurs during pregnancy at greater than 28 weeks gestation
- women with recurrent herpes who are pregnant should be treated with suppressive therapy and be advised that the risk of transmission to their baby is low



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Pap smear. Multinucleated giant cells representing infection by the herpes simplex virus. Note the 3 M's; Multinucleation, Margination of the chromatin, Molding of the nuclei

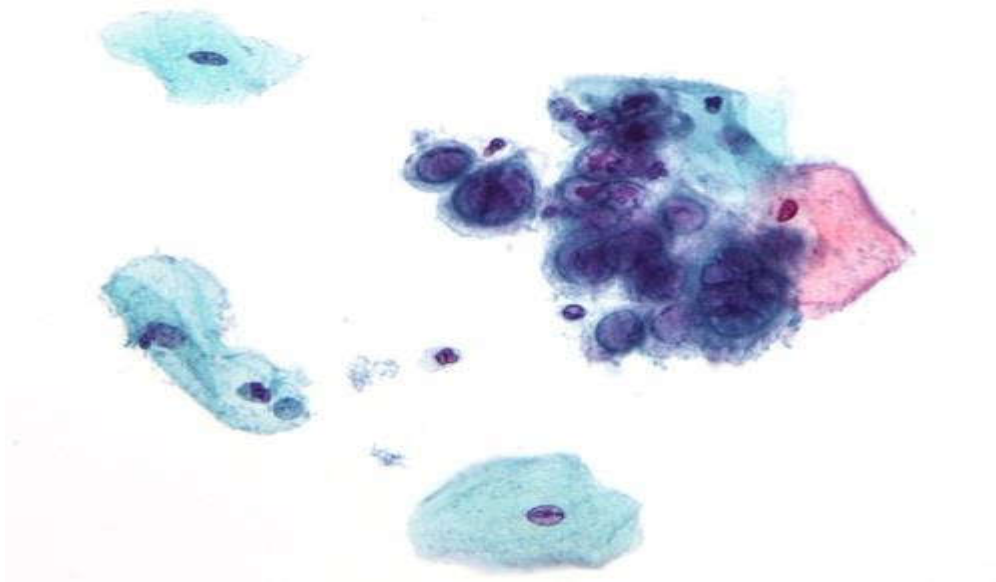


Image sourced from [Wikipedia](#)© Image used on license from PathoPic 🔍

Further Pap smear showing the cytopathic effect of HSV (multi-nucleation, ground glass & margined chromatin)

External Links

[RCOG](#)

2014 Management of Genital Herpes in Pregnancy

Hirsutism and hypertrichosis

/hirsutism is often used to describe androgen-dependent hair growth in women, with hypertrichosis being used for androgen-independent hair growth

Polycystic ovarian syndrome is the most common causes of hirsutism. **Other causes include:**

- Cushing's syndrome
- congenital adrenal hyperplasia
- androgen therapy

Chapter: Dermatology

- obesity: due to peripheral conversion oestrogens to androgens
- adrenal tumour
- androgen secreting ovarian tumour
- drugs: phenytoin.

Assessment of hirsutism

- Ferriman-Gallwey scoring system: 9 body areas are assigned a score of 0 - 4, a score > 15 is considered to indicate moderate or severe hirsutism

Management of hirsutism

- advise weight loss if overweight
- cosmetic techniques such as waxing/bleaching - not available on the NHS
- consider using combined oral contraceptive pills such as co-cyprindiol (Dianette) or ethinylestradiol and drospirenone (Yasmin). Co-cyprindiol should not be used long-term due to the increased risk of venous thromboembolism
- facial hirsutism: topical eflornithine - contraindicated in pregnancy and breast-feeding

Causes of hypertrichosis

- drugs: minoxidil, ciclosporin, diazoxide
- congenital hypertrichosis lanuginosa, congenital hypertrichosis terminalis
- porphyria cutanea tarda
- anorexia nervosa

External Links

[Clinical Knowledge Summaries](#)

Hirsutism

[DermNet NZ](#)

Hirsutism

Hyperhidrosis

Hyperhidrosis describes the excessive production of sweat

Management options include

- topical aluminium chloride preparations are first-line. Main side effect is skin irritation
- iontophoresis: particularly useful for patients with palmar, plantar and axillary hyperhidrosis
- botulinum toxin: currently licensed for axillary symptoms
- surgery: e.g. Endoscopic transthoracic sympathectomy. Patients should be made aware of the risk of compensatory sweating

External Links

[Clinical Knowledge Summaries](#)

Hyperhidrosis guidelines

Impetigo

Impetigo is a superficial bacterial skin infection usually caused by either *Staphylococcus aureus* or *Streptococcus pyogenes*.

Features

- 'golden', crusted skin lesions typically found around the mouth
- very contagious

Management

Limited, localised disease

- topical fusidic acid is first-line
- topical retapamulin is used second-line if fusidic acid has been ineffective or is not tolerated
- MRSA is not susceptible to either fusidic acid or retapamulin. Topical mupirocin (Bactroban) should therefore be used in this situation

Extensive disease

- oral flucloxacillin
- oral erythromycin if penicillin allergic



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[Clinical Knowledge Summaries](#)

Impetigo guidelines

Isotretinoin

Isotretinoin is an oral retinoid used in the treatment of severe acne. Two-thirds of patients have a long term remission or cure following a course of oral isotretinoin

Adverse effects

- teratogenicity: females should ideally be using two forms of contraception (e.g. Combined oral contraceptive pill and condoms)
- dry skin, eyes and lips: the most common side-effect of isotretinoin
- low mood*
- raised triglycerides
- hair thinning
- nose bleeds (caused by dryness of the nasal mucosa)
- benign intracranial hypertension: isotretinoin treatment should not be combined with tetracyclines for this reason
- photosensitivity

*whilst this is a controversial topic, depression and other psychiatric problems are listed in the BNF

External Links

[BNF](#)

Isotretinoin

Keloid scars

Keloid scars are tumour-like lesions that arise from the connective tissue of a scar and extend beyond the dimensions of the original wound

Predisposing factors

- ethnicity: more common in people with dark skin
- occur more commonly in young adults, rare in the elderly
- common sites (in order of decreasing frequency): sternum, shoulder, neck, face, extensor surface of limbs, trunk

Keloid scars are less likely if incisions are made along relaxed skin tension lines*

Treatment

- early keloids may be treated with intra-lesional steroids e.g. triamcinolone
- excision is sometimes required

*Langer lines were historically used to determine the optimal incision line. They were based on procedures done on cadavers but have been shown to produce worse cosmetic results than when following skin tension lines

Keratoacanthoma

Keratoacanthoma is a benign epithelial tumour. They are more frequent in middle age and do not become more common in old age (unlike basal cell and squamous cell carcinoma)

Features - said to look like a volcano or crater

- initially a smooth dome-shaped papule
- rapidly grows to become a crater centrally-filled with keratin

Spontaneous regression of keratoacanthoma within 3 months is common, often resulting in a scar. Such lesions should however be urgently excised as it is difficult clinically to exclude squamous cell carcinoma. Removal also may prevent scarring.



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External Links

[DermNet NZ](https://www.dermnetnz.org/)

Keratoacanthoma pictures

Koebner phenomenon

The Koebner phenomenon describes skin lesions which appear at the site of injury. **It is seen in:**

- psoriasis
- vitiligo
- warts
- lichen planus
- lichen sclerosis
- molluscum contagiosum

External Links

[DermNet NZ](https://www.dermnetnz.org/)

Koebner phenonmen

Lentigo maligna

Lentigo maligna is a type of melanoma in-situ. It typically progresses slowly but may at some stage become invasive causing lentigo maligna melanoma.

Lichen planus

Lichen planus is a skin disorder of unknown aetiology, most probably being immune mediated.

Features

- itchy, papular rash most common on the palms, soles, genitalia and flexor surfaces of arms
- rash often polygonal in shape, 'white-lace' pattern on the surface (Wickham's striae)
- Koebner phenomenon may be seen (new skin lesions appearing at the site of trauma)
- oral involvement in around 50% of patients
- nails: thinning of nail plate, longitudinal ridging



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Chapter: Dermatology

Lichenoid drug eruptions - causes:

- gold
- quinine
- thiazides

Management

- topical steroids are the mainstay of treatment
- extensive lichen planus may require oral steroids or immunosuppression



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Lichen planus

Lichen sclerosis

Lichen sclerosis was previously termed lichen sclerosus et atrophicus. It is an inflammatory condition which usually affects the genitalia and is more common in elderly females. Lichen sclerosis leads to atrophy of the epidermis with white plaques forming

Features

- itch is prominent

The diagnosis is usually made on clinical grounds but a biopsy may be performed if atypical features are present*

Management

- topical steroids and emollients

Follow-up:

- increased risk of vulval cancer

*the RCOG advise the following

*Skin biopsy is not necessary when a diagnosis can be made on clinical examination. **Biopsy is required if the woman fails to respond to treatment or there is clinical suspicion of VIN or cancer.***

and the British Association of Dermatologists state the following:

*A confirmatory biopsy, although ideal, is not always practical, particularly in children. It is not always essential when the clinical features are typical. However, histological examination is advisable if there are atypical features or diagnostic uncertainty and is mandatory if there is any suspicion of neoplastic change. **Patients under routine follow-up will need a biopsy if:***

- *(i) there is a suspicion of neoplastic change, i.e. a persistent area of hyperkeratosis, erosion or erythema, or new warty or papular lesions;*
- *(ii) the disease fails to respond to adequate treatment;*
- *(iii) there is extragenital LS, with features suggesting an overlap with morphea;*
- *(iv) there are pigmented areas, in order to exclude an abnormal melanocytic proliferation;*

and

- *(v) second-line therapy is to be used.*

External Links

[British Association for Sexual Health and HIV](#)

2014 UK National Guideline on the Management of Vulval Conditions

[British Association of Dermatologists](#)

2010 Lichen sclerosus guidelines

[DermNet NZ](#)

Lichen sclerosus

Malignant melanoma: prognostic factors

The invasion depth of a tumour (Breslow depth) is the single most important factor in determining prognosis of patients with malignant melanoma

Breslow Thickness	Approximate 5 year survival
< 1 mm	95-100%
1 - 2 mm	80-96%
2.1 - 4 mm	60-75%
> 4 mm	50%

External Links

[Royal College of Physicians](#)

2012 Advances in the treatment of melanoma

Molluscum contagiosum

◆Molluscum contagiosum is a common skin infection caused by molluscum contagiosum virus (MCV), a member of the Poxviridae family. Transmission occurs directly by close personal contact, or indirectly via fomites (contaminated surfaces) such as shared towels and flannels. The majority of cases occur in children (often in children with atopic eczema), with the maximum incidence in preschool children aged 1-4 years.

◆Typically, molluscum contagiosum presents with characteristic pinkish or pearly white papules with a central umbilication, which are up to 5 mm in diameter. Lesions appear in clusters in areas anywhere on the body (except the palms of the hands and the soles of the feet). In children, lesions are commonly seen on the trunk and in flexures, but anogenital lesions may also occur. In adults, sexual contact may lead to lesions developing on the genitalia, pubis, thighs, and lower abdomen. Rarely, lesions can occur on the oral mucosa and on the eyelids.

Self-care advice:

- Reassure people that molluscum contagiosum is a self-limiting condition.
- Spontaneous resolution usually occurs within 18 months
- Explain that lesions are contagious, and it is sensible to avoid sharing towels, clothing, and baths with uninfected people (e.g. siblings)
- Encourage people not to scratch the lesions. If it is problematic, consider treatment to alleviate the itch
- Exclusion from school, gym, or swimming is not necessary

Treatment is not usually recommended. If lesions are troublesome or considered unsightly, use simple trauma or cryotherapy, depending on the parents' wishes and the child's age:

- **Squeezing** (with fingernails) or piercing (orange stick) lesions may be tried, following a bath. Treatment should be limited to a few lesions at one time
- **Cryotherapy** may be used in older children or adults, if the healthcare professional is experienced in the procedure
- **Eczema** or inflammation can develop around lesions prior to resolution. Treatment may be required if:
 - Itching is problematic; prescribe an emollient and a mild topical corticosteroid (e.g. hydrocortisone 1%)
 - The skin looks infected (e.g. oedema, crusting); prescribe a topical antibiotic (e.g. fusidic acid 2%)

Referral may be necessary in some circumstances:

- For people who are HIV-positive with extensive lesions urgent referral to a HIV specialist
- For people with eyelid-margin or ocular lesions and associated red eye urgent referral to an ophthalmologist
- Adults with anogenital lesions should be referred to genito-urinary medicine, for screening for other sexually transmitted infections



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Mycosis fungoides

Mycosis fungoides is a rare form of T-cell lymphoma that affects the skin.

Features

- itchy, red patches which are typically confused with eczema or psoriasis initially
- lesions tend to be of different colours in contrast to eczema/psoriasis where there is greater homogeneity



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Image showing the plaque stage of mycosis fungoides



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Further image showing the plaque stage of mycosis fungoides

Nickel dermatitis

Nickel is a common cause allergic contact dermatitis and is an example of a type IV hypersensitivity reaction. It is often caused by jewellery such as watches

It is diagnosed by a skin patch test

Nickel dermatitis

Nickel is a common cause allergic contact dermatitis and is an example of a type IV hypersensitivity reaction. It is often caused by jewellery such as watches

It is diagnosed by a skin patch test

Onycholysis

Onycholysis describes the separation of the nail plate from the nail bed

Causes

- idiopathic
 - trauma e.g. Excessive manicuring
 - infection: especially fungal
 - skin disease: psoriasis, dermatitis
 - impaired peripheral circulation e.g. Raynaud's
 - systemic disease: hyper- and hypothyroidism
-

External Links

DermIS.net

Picture of onycholysis

Otitis externa

Otitis externa is a common reason for primary care attendance in the UK.

Causes of otitis externa include:

- infection: bacterial (*Staphylococcus aureus*, *Pseudomonas aeruginosa*) or fungal
- seborrhoeic dermatitis
- contact dermatitis (allergic and irritant)

Features

- ear pain, itch, discharge
- otoscopy: red, swollen, or eczematous canal

The recommend initial management of otitis externa is:

- topical antibiotic or a combined topical antibiotic with steroid
- if the tympanic membrane is perforated aminoglycosides are traditionally not used*
- if there is canal debris then consider removal
- if the canal is extensively swollen then an ear wick is sometimes inserted

Second line options include:

- consider contact dermatitis secondary to neomycin
- oral antibiotics if the infection is spreading
- taking a swab inside the ear canal
- empirical use of an antifungal agent

Malignant otitis externa is more common in elderly diabetics. In this condition there is extension of infection into the bony ear canal and the soft tissues deep to the bony canal. Intravenous antibiotics may be required.

*many ENT doctors disagree with this and feel that concerns about ototoxicity are unfounded

External Links

[Clinical Knowledge Summaries](#)

Otitis externa guidelines

Parvovirus B19

Parvovirus B19 is a DNA virus which causes a variety of clinical presentations. It was identified in the 1980's as the cause of erythema infectiosum

Erythema infectiosum (also known as fifth disease or 'slapped-cheek syndrome'):

- most common presenting illness
- systemic symptoms: lethargy, fever, headache
- 'slapped-cheek' rash spreading to proximal arms and extensor surfaces

Other presentations

- asymptomatic
- pancytopenia in immunosuppressed patients
- aplastic crises e.g. in sickle-cell disease (parvovirus B19 suppresses erythropoiesis for about a week so aplastic anaemia is rare unless there is a chronic haemolytic anaemia)

External Links

[DermNet NZ](#)

Fifth disease

Pellagra

Pellagra is caused by nicotinic acid (niacin) deficiency. The classical features are the 3 D's - dermatitis, diarrhoea and dementia

Pellagra may occur as a consequence of isoniazid therapy (isoniazid inhibits the conversion of tryptophan to niacin) and it is more common in alcoholics.

Features

- dermatitis (brown scaly rash on sun-exposed sites - termed Casal's necklace if around neck)
- diarrhoea
- dementia, depression
- death if not treated

External Links

[DermNet NZ](#)

Pellagra

Pemphigus vulgaris

Pemphigus vulgaris is an autoimmune disease caused by antibodies directed against desmoglein 3, a cadherin-type epithelial cell adhesion molecule. It is more common in the Ashkenazi Jewish population

Features

- mucosal ulceration is common and often the presenting symptom. Oral involvement is seen in 50-70% of patients
- skin blistering - flaccid, easily ruptured vesicles and bullae. Lesions are typically painful but not itchy. These may develop months after the initial mucosal symptoms. Nikolsky's describes the spread of bullae following application of horizontal, tangential pressure to the skin
- acantholysis on biopsy



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Mucosal ulceration is common with pemphigus



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Management

- steroids
- immunosuppressants

External Links

[DermNet NZ](https://www.dermnetnz.org/)

Pemphigus vulgaris

Pityriasis rosea

Pityriasis rosea describes an acute, self-limiting rash which tends to affect young adults. The aetiology is not fully understood but is thought that herpes hominis virus 7 (HHV-7) may play a role.

Features

- herald patch (usually on trunk)
- followed by erythematous, oval, scaly patches which follow a characteristic distribution with the longitudinal diameters of the oval lesions running parallel to the line of Langer. This may produce a 'fir-tree' appearance

Management

- self-limiting, usually disappears after 4-12 weeks



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On the left a typical herald patch is seen. After a few days a more generalised 'fir-tree' rash appears



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Differentiating guttate psoriasis and pityriasis rosea

	Guttate psoriasis	Pityriasis rosea
Prodrome	Classically preceded by a streptococcal sore throat 2-4 weeks	Many patients report recent respiratory tract infections but this is not common in questions
Appearance	'Tear drop', scaly papules on the trunk and limbs	Herald patch followed 1-2 weeks later by multiple erythematous, slightly raised oval lesions with a fine scale confined to the outer aspects of the lesions. May follow a characteristic distribution with the longitudinal diameters of the oval lesions running parallel to the line of Langer. This may produce a 'fir-tree' appearance
Treatment / natural history	Most cases resolve spontaneously within 2-3 months Topical agents as per psoriasis UVB phototherapy	Self-limiting, resolves after around 6 weeks

External Links

[DermNet NZ](#)

Pityriasis rosea

Pityriasis versicolor

Pityriasis versicolor, also called tinea versicolor, is a superficial cutaneous fungal infection caused by *Malassezia furfur* (formerly termed *Pityrosporum ovale*)

Features

- most commonly affects trunk
- patches may be hypopigmented, pink or brown (hence versicolor). May be more noticeable following a suntan
- scale is common
- mild pruritus

Predisposing factors

- occurs in healthy individuals
- immunosuppression
- malnutrition
- Cushing's

Management

- topical antifungal. NICE Clinical Knowledge Summaries advise ketoconazole shampoo as this is more cost effective for large areas
- if extensive disease or failure to respond to topical treatment then consider oral itraconazole

External Links

[DermNet NZ](#)

Pityriasis versicolor

Polycystic ovarian syndrome: management

Polycystic ovarian syndrome (PCOS) is a complex condition of ovarian dysfunction thought to affect between 5-20% of women of reproductive age. Management is complicated and problem based partly because the aetiology of PCOS is not fully understood. Both hyperinsulinaemia and high levels of luteinizing hormone are seen in PCOS and there appears to be some overlap with the metabolic syndrome.

General

- weight reduction if appropriate
- if a woman requires contraception then a combined oral contraceptive (COC) pill may help regulate her cycle and induce a monthly bleed (see below)

Hirsutism and acne

- a COC pill may be used help manage hirsutism. Possible options include a third generation COC which has fewer androgenic effects or co-cyprindiol which has an anti-androgen action. Both of these types of COC may carry an increased risk of venous thromboembolism
- if doesn't respond to COC then topical eflornithine may be tried
- spironolactone, flutamide and finasteride may be used under specialist supervision

Infertility

- weight reduction if appropriate
- the management of infertility in patients with PCOS should be supervised by a specialist. There is an ongoing debate as to whether metformin, clomifene or a combination should be used to stimulate ovulation
- a 2007 trial published in the New England Journal of Medicine suggested clomifene was the most effective treatment. There is a potential risk of multiple pregnancies with anti-oestrogen* therapies such as clomifene. The RCOG published an opinion paper in 2008 and concluded that on current evidence metformin is not a first line treatment of choice in the management of PCOS
- metformin is also used, either combined with clomifene or alone, particularly in patients who are obese
- gonadotrophins

*work by occupying hypothalamic oestrogen receptors without activating them. This interferes with the binding of oestradiol and thus prevents negative feedback inhibition of FSH secretion

External Links

[NEJM](#)

Summary of recent trial comparing metformin to clomifene

[RCOG](#)

Opinion paper on metformin in PCOS

[Clinical Knowledge Summaries](#)

PCOS management

Pompholyx

Pompholyx is a type of eczema which affects both the hands (cheiropompholyx) and the feet (pedopompholyx). It is also known as dyshidrotic eczema

Features

- small blisters on the palms and soles
- pruritic, sometimes burning sensation
- once blisters burst skin may become dry and crack

Management

- cool compresses
- emollients
- topical steroids

External Links

[Dermnet NZ](#)

Photos of pompholyx

Porphyria cutanea tarda

Porphyria cutanea tarda is the most common hepatic porphyria. It is due to an inherited defect in uroporphyrinogen decarboxylase or caused by hepatocyte damage e.g. alcohol, hepatitis C, oestrogens

Features

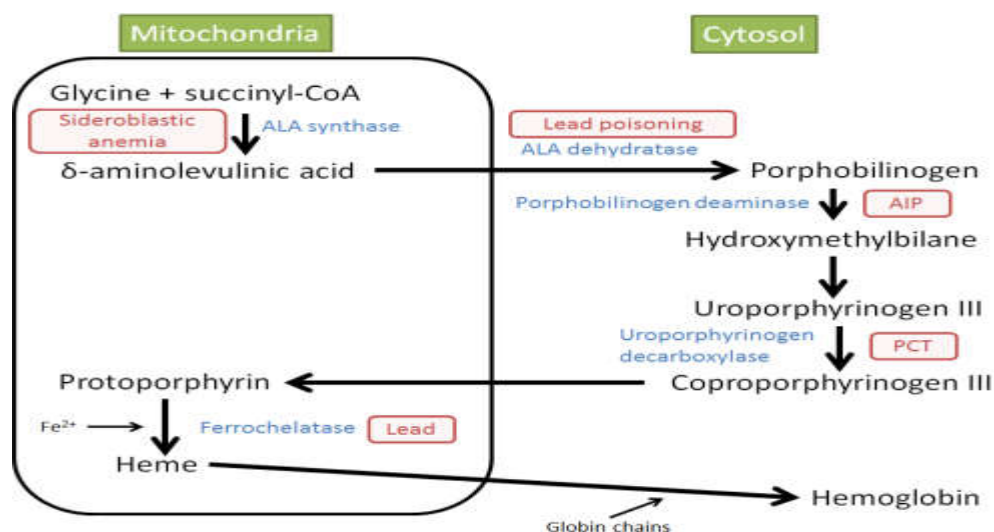
- classically presents with photosensitive rash with blistering and skin fragility on the face and dorsal aspect of hands (most common feature)
- hypertrichosis
- hyperpigmentation

Investigations

- urine: elevated uroporphyrinogen and pink fluorescence of urine under Wood's lamp

Management

- chloroquine
- venesection



External Links

[DermNet NZ](#)

Porphyria cutanea tarda

Pressure ulcers

The following is based on a 2009 NHS Best Practice Statement. Please see the link for further details. Some selected points are listed below. NICE also published guidelines in 2014.

Pressure ulcers develop in patients who are unable to move parts of their body due to illness, paralysis or advancing age. They typically develop over bony prominences such as the sacrum or heel. **The following factors predispose to the development of pressure ulcers:**

- malnourishment
- incontinence
- lack of mobility
- pain (leads to a reduction in mobility)

The **Waterlow score** is widely used to screen for patients who are at risk of developing pressure areas. It includes a number of factors including body mass index, nutritional status, skin type, mobility and continence.

Grading of pressure ulcers - the following is taken from the European Pressure Ulcer Advisory Panel classification system.

Grade	Findings
Grade 1	Non-blanchable erythema of intact skin. Discolouration of the skin, warmth, oedema, induration or hardness may also be used as indicators, particularly on individuals with darker skin
Grade 2	Partial thickness skin loss involving epidermis or dermis, or both. The ulcer is superficial and presents clinically as an abrasion or blister
Grade 3	Full thickness skin loss involving damage to or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia.
Grade 4	Extensive destruction, tissue necrosis, or damage to muscle, bone or supporting structures with or without full thickness skin loss

Management

- a moist wound environment encourages ulcer healing. Hydrocolloid dressings and hydrogels may help facilitate this. The use of soap should be discouraged to avoid drying the wound
- wound swabs should not be done routinely as the vast majority of pressure ulcers are colonised with bacteria. The decision to use systemic antibiotics should be taken on a clinical basis (e.g. Evidence of surrounding cellulitis)
- consider referral to the tissue viability nurse
- surgical debridement may be beneficial for selected wounds

External Links

[NICE](#)

2014 The prevention and treatment of pressure ulcers

[NHS](#)

Prevention and management of pressure ulcers

Pruritus

The table below lists the main characteristics of the most important causes of pruritus

Liver disease	History of alcohol excess Stigmata of chronic liver disease: spider naevi, bruising, palmar erythema, gynaecomastia etc Evidence of decompensation: ascites, jaundice, encephalopathy
Iron deficiency anaemia	Pallor Other signs: koilonychia, atrophic glossitis, post-cricoid webs, angular stomatitis
Polycythaemia	Pruritus particularly after warm bath 'Ruddy complexion' Gout Peptic ulcer disease
Chronic kidney disease	Lethargy & pallor Oedema & weight gain Hypertension
Lymphoma	Night sweats Lymphadenopathy Splenomegaly, hepatomegaly Fatigue

Other causes:

- hyper- and hypothyroidism
- diabetes
- pregnancy
- 'senile' pruritus
- urticaria
- skin disorders: eczema, scabies, psoriasis, pityriasis rosea

Psoriasis

Psoriasis is a common (prevalence around 2%) and chronic skin disorder. It generally presents with red, scaly patches on the skin although it is now recognised that patients with psoriasis are at increased risk of arthritis and cardiovascular disease.

Pathophysiology

- multifactorial and not yet fully understood
- genetic: associated HLA-B13, -B17, and -Cw6. Strong concordance (70%) in identical twins
- immunological: abnormal T cell activity stimulates keratinocyte proliferation. There is increasing evidence this may be mediated by a novel group of T helper cells producing IL-17, designated Th17. These cells seem to be a third T-effector cell subset in addition to Th1 and Th2
- environmental: it is recognised that psoriasis may be worsened (e.g. Skin trauma, stress), triggered (e.g. Streptococcal infection) or improved (e.g. Sunlight) by environmental factors

Recognised subtypes of psoriasis

- plaque psoriasis: the most common sub-type resulting in the typical well demarcated red, scaly patches affecting the extensor surfaces, sacrum and scalp
- **flexural psoriasis**: in contrast to plaque psoriasis the skin is smooth
- guttate psoriasis: transient psoriatic rash frequently triggered by a streptococcal infection. Multiple red, teardrop lesions appear on the body
- pustular psoriasis: commonly occurs on the palms and soles



Other features

- nail signs: pitting, onycholysis
- arthritis

Complications

- psoriatic arthropathy (around 10%)
- increased incidence of metabolic syndrome
- increased incidence of cardiovascular disease
- increased incidence of venous thromboembolism
- psychological distress



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External Links

[SIGN](#)

2010 Psoriasis guidelines

[DermNet NZ](#)

Scalp psoriasis

Psoriasis: exacerbating factors

The following factors may exacerbate psoriasis:

- trauma
- alcohol
- drugs: beta blockers, lithium, antimalarials (chloroquine and hydroxychloroquine), NSAIDs and ACE inhibitors, infliximab
- withdrawal of systemic steroids

External Links

[DermNet NZ](#)

Psoriasis

Psoriasis: guttate

Guttate psoriasis is more common in children and adolescents. It may be precipitated by a streptococcal infection 2-4 weeks prior to the lesions appearing

Features

- tear drop papules on the trunk and limbs



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Management

- most cases resolve spontaneously within 2-3 months
- there is no firm evidence to support the use of antibiotics to eradicate streptococcal infection
- topical agents as per psoriasis
- UVB phototherapy
- tonsillectomy may be necessary with recurrent episodes.

Differentiating guttate psoriasis and pityriasis rosea

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External Links

[Clinical Knowledge Summaries](#)

Guttate psoriasis guidelines

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Guttate psoriasis

Psoriasis: management

NICE released guidelines in 2012 on the management of psoriasis and psoriatic arthropathy. Please see the link for more details.

Management of chronic plaque psoriasis

- regular emollients may help to reduce scale loss and reduce pruritus
- first-line: NICE recommend a potent corticosteroid applied once daily plus vitamin D analogue applied once daily (applied separately, one in the morning and the other in the evening) for up to 4 weeks as initial treatment
- second-line: if no improvement after 8 weeks then offer a vitamin D analogue twice daily
- third-line: if no improvement after 8-12 weeks then offer either: a potent corticosteroid applied twice daily for up to 4 weeks or a coal tar preparation applied once or twice daily
- short-acting dithranol can also be used



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Using topical steroids in psoriasis

- as we know topical corticosteroid therapy may lead to skin atrophy, striae and rebound symptoms
- systemic side-effects may be seen when potent corticosteroids are used on large areas e.g. > 10% of the body surface area
- NICE recommend that we aim for a 4 week break before starting another course of topical corticosteroids
- they also recommend using potent corticosteroids for no longer than 8 weeks at a time and very potent corticosteroids for no longer than 4 weeks at a time

What should I know about vitamin D analogues?

- examples of vitamin D analogues include calcipotriol (Dovonex), calcitriol and tacalcitol
- they work by reducing cell division and differentiation
- adverse effects are uncommon
- unlike corticosteroids they may be used long-term
- unlike coal tar and dithranol they do not smell or stain
- they tend to reduce the scale and thickness of plaques but not the erythema
- they should be avoided in pregnancy
- the maximum weekly amount for adults is 100g



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A 'before and after' image showing the effect of 6 weeks of calcipotriol therapy on a large plaque. Note how the scale has improved but the erythema remains

Steroids in psoriasis

- topical steroids are commonly used in flexural psoriasis and there is also a role for mild steroids in facial psoriasis. If steroids are ineffective for these conditions vitamin D analogues or tacrolimus ointment should be used second line
- patients should have 4 week breaks between course of topical steroids
- very potent steroids should not be used for longer than 4 weeks at a time. Potent steroids can be used for up to 8 weeks at a time
- the scalp, face and flexures are particularly prone to steroid atrophy so topical steroids should not be used for more than 1-2 weeks/month.

Scalp psoriasis

- NICE recommend the use of potent topical corticosteroids used once daily for 4 weeks
- if no improvement after 4 weeks then either use a different formulation of the potent corticosteroid (for example, a shampoo or mousse) and/or a topical agents to remove adherent scale (for example, agents containing salicylic acid, emollients and oils) before application of the potent corticosteroid

Face, flexural and genital psoriasis

- NICE recommend offering a mild or moderate potency corticosteroid applied once or twice daily for a maximum of 2 weeks

Secondary care management

Phototherapy

- narrow band ultraviolet B light is now the treatment of choice. If possible this should be given 3 times a week
- photochemotherapy is also used - psoralen + ultraviolet A light (PUVA)
- adverse effects: skin ageing, squamous cell cancer (not melanoma)

Systemic therapy

- oral methotrexate is used first-line. It is particularly useful if there is associated joint disease
- ciclosporin
- systemic retinoids
- biological agents: infliximab, etanercept and adalimumab
- ustekinumab (IL-12 and IL-23 blocker) is showing promise in early trials

Mechanism of action of commonly used drugs:

- **coal tar**: probably inhibit DNA synthesis
- **calcipotriol**: vitamin D analogue which reduces epidermal proliferation and restores a normal horny layer
- **dithranol**: inhibits DNA synthesis, wash off after 30 mins, SE: burning, staining

External Links

[NICE](#)

2012 Psoriasis guidelines

[SIGN](#)

2010 Psoriasis guidelines

Pyoderma gangrenosum

Features

- typically on the lower limbs
- initially small red papule
- later deep, red, necrotic ulcers with a violaceous border
- may be accompanied systemic symptoms e.g. Fever, myalgia

Causes*

- idiopathic in 50%
- inflammatory bowel disease: ulcerative colitis, Crohn's
- rheumatoid arthritis, SLE
- myeloproliferative disorders
- lymphoma, myeloid leukaemias
- monoclonal gammopathy (IgA)
- primary biliary cirrhosis

Management

- the potential for rapid progression is high in most patients and most doctors advocate oral steroids as first-line treatment
- other immunosuppressive therapy, for example ciclosporin and infliximab, have a role in difficult cases

*note whilst pyoderma gangrenosum can occur in diabetes mellitus it is rare and is generally not included in a differential of potential causes



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Pyoderma gangrenosum

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Stoma skin problems

Pyogenic granuloma

Pyogenic granuloma is a relatively common benign skin lesion. The name is confusing as they are neither true granulomas nor pyogenic in nature. There are multiple alternative names but perhaps 'eruptive haemangioma' is the most useful.

The cause of pyogenic granuloma is not known but a number of factors are linked:

- trauma
- pregnancy
- more common in women and young adults

Features

- most common sites are head/neck, upper trunk and hands. Lesions in the oral mucosa are common in pregnancy
- initially small red/brown spot
- rapidly progress within days to weeks forming raised, red/brown lesions which are often spherical in shape
- the lesions may bleed profusely or ulcerate

Management

- lesions associated with pregnancy often resolve spontaneously post-partum
- other lesions usually persist. Removal methods include curettage and cauterisation, cryotherapy, excision



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Pyogenic granuloma

Sarcoidosis

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia
- insidious: dyspnoea, non-productive cough, malaise, weight loss
- skin: lupus pernio
- hypercalcaemia: macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol)

Syndromes associated with sarcoidosis

Lofgren's syndrome is an acute form of the disease characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia. It usually carries an excellent prognosis

In **Mikulicz syndrome*** there is enlargement of the parotid and lacrimal glands due to sarcoidosis, tuberculosis or lymphoma

Heerfordt's syndrome (uveoparotid fever) there is parotid enlargement, fever and uveitis secondary to sarcoidosis

*this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome

Seborrhoeic dermatitis in adults

Seborrhoeic dermatitis in adults is a chronic dermatitis thought to be caused by an inflammatory reaction related to a proliferation of a normal skin inhabitant, a fungus called *Malassezia furfur* (formerly known as *Pityrosporum ovale*). It is common, affecting around 2% of the general population

Features

- eczematous lesions on the sebum-rich areas: scalp (may cause dandruff), periorbital, auricular and nasolabial folds
- otitis externa and blepharitis may develop

Chapter: Dermatology

Associated conditions include:

- HIV
- Parkinson's disease

Scalp disease management

- over the counter preparations containing zinc pyrithione ('Head & Shoulders') and tar ('Neutrogena T/Gel') are first-line
- the preferred second-line agent is ketoconazole
- selenium sulphide and topical corticosteroid may also be useful

Face and body management

- topical antifungals: e.g. Ketoconazole
- topical steroids: best used for short periods
- difficult to treat - recurrences are common

External Links

[Clinical Knowledge Summaries](#)

Seborrhoeic dermatitis guidelines

[DermNet NZ](#)

Overview and pictures of seborrhoeic dermatitis

Sezary syndrome

Sezary syndrome is a type of T-cell cutaneous lymphoma.

Features

- pruritus
- erythroderma typically affecting the palms, soles and face
- atypical T cells
- lymphadenopathy
- hepatosplenomegaly

External Links

[Royal College of Physicians](#)

2012 Management of cutaneous T-cell lymphoma

Shin lesions

The differential diagnosis of shin lesions includes the following conditions:

- erythema nodosum
- pretibial myxoedema
- pyoderma gangrenosum
- necrobiosis lipoidica diabetorum

Below are the characteristic features:

Erythema nodosum

- symmetrical, erythematous, tender, nodules which heal without scarring
- most common causes are streptococcal infections, sarcoidosis, inflammatory bowel disease and drugs (penicillins, sulphonamides, oral contraceptive pill)

Pretibial myxoedema

- symmetrical, erythematous lesions seen in Graves' disease
- shiny, orange peel skin

Pyoderma gangrenosum

- initially small red papule
- later deep, red, necrotic ulcers with a violaceous border
- idiopathic in 50%, may also be seen in inflammatory bowel disease, connective tissue disorders and myeloproliferative disorders

Necrobiosis lipoidica diabetorum

- shiny, painless areas of yellow/red skin typically on the shin of diabetics
- often associated with telangiectasia

External Links

[DermNet NZ](#)

Erythema nodosum

[DermIS.net](#)

Picture of pretibial myxoedema

[DermNet NZ](#)

Pyoderma gangrenosum

[DermNet NZ](#)

Necrobiosis lipoidica

Skin disorders associated with diabetes

Note whilst pyoderma gangrenosum can occur in diabetes mellitus it is rare and is often not included in a differential of potential causes

Necrobiosis lipoidica

- shiny, painless areas of yellow/red/brown skin typically on the shin
- often associated with surrounding telangiectasia

Infection

- candidiasis
- staphylococcal

Neuropathic ulcers

Vitiligo

Lipoatrophy

Granuloma annulare*

- papular lesions that are often slightly hyperpigmented and depressed centrally

*it is not clear from recent studies if there is actually a significant association between diabetes mellitus and granuloma annulare, but it is often listed in major textbooks

External Links

[DermNet NZ](#)

Necrobiosis lipoidic

[DermNet NZ](#)

Granuloma annulare

Skin disorders associated with pregnancy

Polymorphic eruption of pregnancy

- pruritic condition associated with last trimester
- lesions often first appear in abdominal striae
- management depends on severity: emollients, mild potency topical steroids and oral steroids may be used



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Polymorphic eruption of pregnancy



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Polymorphic eruption of pregnancy



Polymorphic eruption of pregnancy

Pemphigoid gestationis

- pruritic blistering lesions
- often develop in peri-umbilical region, later spreading to the trunk, back, buttocks and arms
- usually presents 2nd or 3rd trimester and is rarely seen in the first pregnancy
- oral corticosteroids are usually required



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Pemphigoid gestationis



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Pemphigoid gestationis

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Polymorphic eruption of pregnancy

[DermNet NZ](https://www.dermnetnz.org/)

Pemphigoid gestationis

Skin disorders associated with tuberculosis

Possible skin disorders

- lupus vulgaris (accounts for 50% of cases)
- erythema nodosum
- scarring alopecia
- scrofuloderma: breakdown of skin overlying a tuberculous focus
- verrucosa cutis
- gumma.

♦Lupus vulgaris is the most common form of cutaneous TB seen in the Indian subcontinent. It generally occurs on the face and is common around the nose and mouth. The initial lesion is an erythematous flat plaque which gradually becomes elevated and may ulcerate later

External Links

[DermNet NZ](#)

Picture of lupus vulgaris

[DermIS.net](#)

Pictures of scrofuloderma

Systemic mastocytosis

Systemic mastocytosis results from a neoplastic proliferation of mast cells

Features

- urticaria pigmentosa - produces a wheal on rubbing (Darier's sign)
- flushing
- abdominal pain
- monocytosis on the blood film

Diagnosis

- raised serum tryptase levels
- urinary histamine

Tinea

Tinea is a term given to dermatophyte fungal infections. **Three main types of infection are described depending on what part of the body is infected:**

- **tinea capitis - scalp**
- **tinea corporis - trunk, legs or arms**
- **tinea pedis - feet**

Tinea capitis (scalp ringworm)

- a cause of scarring alopecia mainly seen in children
- if untreated a raised, pustular, spongy/boggy mass called a kerion may form
- most common cause is *Trichophyton tonsurans* in the UK and the USA
- may also be caused by *Microsporum canis* acquired from cats or dogs
- **diagnosis:** lesions due to *Microsporum canis* green fluorescence under Wood's lamp*. However the most useful investigation is scalp scrapings.

Chapter: Dermatology

- **management** (based on CKS guidelines): oral antifungals: terbinafine for *Trichophyton tonsurans* infections and griseofulvin for *Microsporum* infections. Topical ketoconazole shampoo should be given for the first two weeks to reduce transmission



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Image showing a kerion

Tinea corporis (ringworm)

- causes include *Trichophyton rubrum* and *Trichophyton verrucosum* (e.g. From contact with cattle)
- well-defined annular, erythematous lesions with pustules and papules
- may be treated with oral fluconazole



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Image showing tinea corporis



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Image showing tinea corporis. Note the well defined border

Chapter: Dermatology

Tinea pedis (athlete's foot)

- characterised by itchy, peeling skin between the toes
- common in adolescence

*lesions due to *Trichophyton* species do not readily fluoresce under Wood's lamp

External Links

[Clinical Knowledge Summaries](#)

Fungal skin infection - scalp

Toxic epidermal necrolysis

Toxic epidermal necrolysis (TEN) is a potentially life-threatening skin disorder that is most commonly seen secondary to a drug reaction. In this condition the skin develops a scalded appearance over an extensive area. Some authors consider TEN to be the severe end of a spectrum of skin disorders which includes erythema multiforme and Stevens-Johnson syndrome

Features

- systemically unwell e.g. pyrexia, tachycardic
- positive Nikolsky's sign: the epidermis separates with mild lateral pressure

Drugs known to induce TEN

- phenytoin
- sulphonamides
- allopurinol
- penicillins
- carbamazepine
- NSAIDs

Management

- stop precipitating factor
- supportive care, often in intensive care unit
- intravenous immunoglobulin has been shown to be effective and is now commonly used first-line
- other treatment options include: immunosuppressive agents (ciclosporin and cyclophosphamide), plasmapheresis

External Links

[DermNet NZ](#)

Toxic epidermal necrolysis

Venous ulceration

Venous ulceration is typically seen above the medial malleolus

Investigations

- ankle-brachial pressure index (ABPI) is important in non-healing ulcers to assess for poor arterial flow which could impair healing
- a 'normal' ABPI may be regarded as between 0.9 - 1.2. Values below 0.9 indicate arterial disease. Interestingly, values above 1.3 may also indicate arterial disease, in the form of false-negative results secondary to arterial calcification (e.g. In diabetics)

Management

- compression bandaging, usually four layer (only treatment shown to be of real benefit)
- oral pentoxifylline, a peripheral vasodilator, improves healing rate
- small evidence base supporting use of flavinoids
- little evidence to suggest benefit from hydrocolloid dressings, topical growth factors, ultrasound therapy and intermittent pneumatic compression

Vitiligo

Vitiligo is an autoimmune condition which results in the loss of melanocytes and consequent depigmentation of the skin. It is thought to affect around 1% of the population and symptoms typically develop by the age of 20-30 years.

Features

- well demarcated patches of depigmented skin
- the peripheries tend to be most affected
- trauma may precipitate new lesions (Koebner phenomenon)

Associated conditions

- type 1 diabetes mellitus
- Addison's disease
- autoimmune thyroid disorders
- pernicious anaemia
- alopecia areata

Management

- sun block for affected areas of skin
- camouflage make-up
- topical corticosteroids may reverse the changes if applied early
- there may also be a role for topical tacrolimus and phototherapy, although caution needs to be exercised with light-skinned patients



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Vitiligo

Yellow nail syndrome

Slowing of the nail growth leads to the characteristic thickened and discoloured nails seen in yellow nail syndrome.

Associations

- congenital lymphoedema
- pleural effusions
- bronchiectasis
- chronic sinus infections

External Links

[Dermnet NZ](#)

Yellow Nail Syndrome

Zinc deficiency

Features

- perioral dermatitis: red, crusted lesions
 - acrodermatitis
 - alopecia
 - short stature
 - hypogonadism
 - hepatosplenomegaly
 - geophagia (ingesting clay/soil)
 - cognitive impairment
-